



Case Report

Hepatic inflammatory pseudotumor mimicking hepatocellular carcinoma

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Abstract

Inflammatory pseudotumor of the liver is a rare tumor. It has variable clinical presentations and image findings. It can mimic benign or malignant hepatic tumors, and may be difficult to diagnose. We present a case in which a hepatic inflammatory pseudotumor was misdiagnosed as hepatocellular carcinoma because the tumor presented a typical enhancing profile and morphology of hepatocellular carcinoma on computed tomography, and the patient had liver cirrhosis. However, a thicker tumor capsule than that of typical hepatocellular carcinoma was noted while reviewing the computed tomography images. A capsule of inflammatory pseudotumor thicker than that of hepatocellular carcinoma has never been reported in the literature before, and could be an important diagnostic clue to differentiate inflammatory pseudotumor from hepatocellular carcinoma.

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1. Introduction

Inflammatory pseudotumor (IPT) is a benign tumor of uncertain cause. It is composed of fibrous tissue, lymphocytes, and plasma cells. The tumor can present in many organs, but the lungs and liver are the most common sites.¹ Various image findings have been reported, some mimicking those of a malignant hepatic tumor. We present a case of hepatic IPT with the typical image findings of hepatocellular carcinoma (HCC) on computed tomography (CT).

2. Case report

An 85-year-old female with history of chronic hepatitis C without treatment and type II diabetes mellitus under medical control visited our outpatient department due to abdominal fullness. Laboratory data showed elevated serum alanine transaminase (200 mg/dL) and alpha-fetoprotein (81.6 mg/dL)

levels, without elevated serum C-reactive protein level, increased erythrocyte sedimentation rate, or leukocytosis. No definite body weight loss had been noticed. Physical examination on admission to our hospital was unremarkable except for a surgical scar from a previous appendectomy.

Sonography revealed a coarse echogenic background to the liver and a hypoechoic mass lesion in the right lobe of the liver measuring 3.5 cm in diameter. Dynamic contrast-enhanced CT of the abdomen revealed liver cirrhosis with a nodular liver surface and a well-defined mass lesion, measuring 3.5 cm in diameter, in segment 5 of the liver, with low density in the precontrast phase, early inhomogeneous enhancement in the arterial phase, and contrast medium washout in the venous phase. A thick, peripheral enhancing tumor capsule measuring 2–3.3 mm in thickness was depicted, more obvious in the venous phase (Fig. 1). No ascites was noted on the CT examination.

According to the practice guideline of American Association for the Study of Liver Disease, HCC was diagnosed preoperatively. Because the patient was otherwise well and was requesting curative treatment, hepatic segmentectomy was performed.

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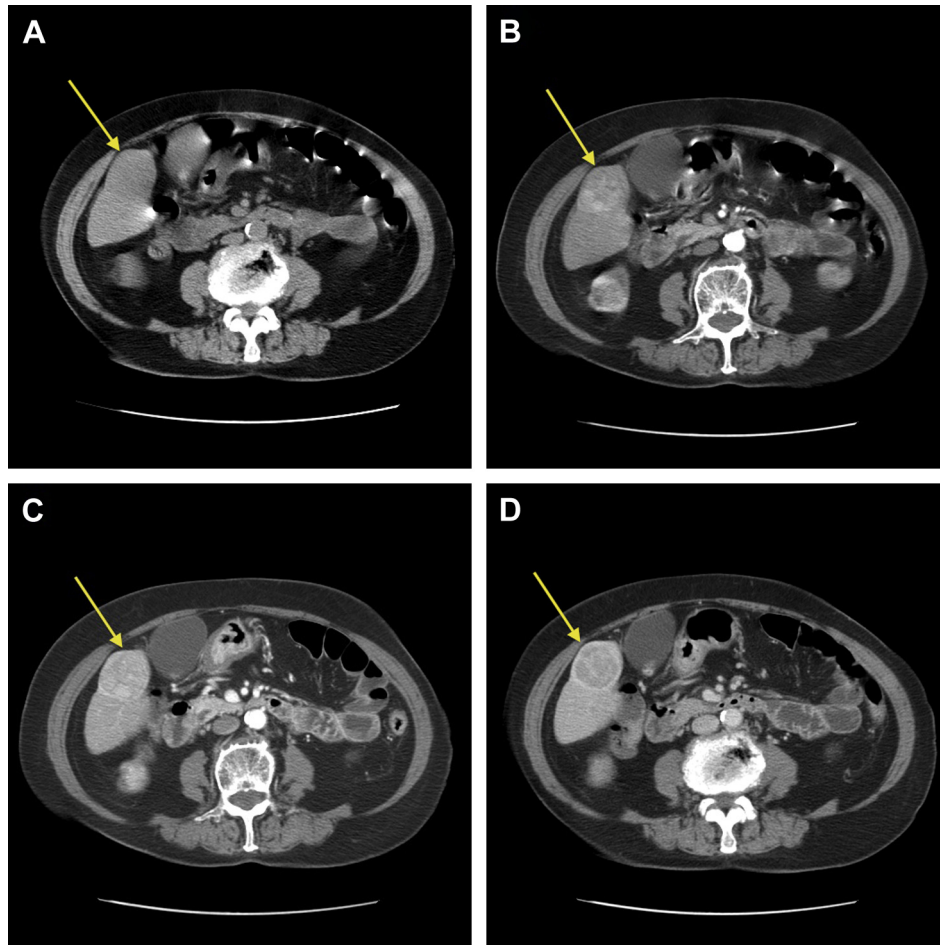


Fig. 1. Dynamic abdominal computed tomography revealed a mildly hypodense lesion (arrowed) in segment 5 of the liver (A), measuring 3.5 cm in diameter, with early enhancement (B,C) and contrast washout in the venous phase (D), and with a hyperdense tumor capsule.

After surgery, histologic examination revealed a well-defined mass with lymphocyte and multinucleated giant cell infiltration, fibrin deposition, and formation of granulation tissue (Fig. 2). Foci of caseating granulomatous inflammation with a necrotic center were surrounded by epithelioid histiocytes. A thick fibrous capsule measuring 2.5 mm in thickness was also depicted. Special staining with

periodic acid–Schiff and an immunohistochemical study for tuberculosis failed to demonstrate a fungal or mycobacterial infection. The final pathologic diagnosis was inflammatory IPT.

After hepatic segmentectomy, the patient was generally well without complications, except for mild ascites identified during outpatient department follow-up, for which diuretics

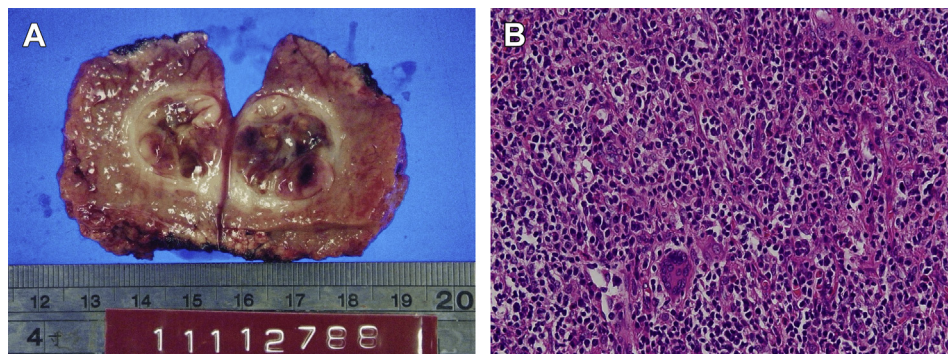


Fig. 2. The surgical specimen (A) showed a mass lesion with inner hemorrhage and a peripheral thick fibrous capsule measuring 2.5 mm in thickness. (B) A photomicrograph (original magnification, 40 \times ; H&E stain) revealed lymphoplasmic cells and multinucleated giant cell infiltration. The final pathologic diagnosis was inflammatory pseudotumor.

were administered. No definite tumor recurrence was seen at follow-up sonography.

3. Discussion

IPT is a benign lesion consisting of proliferating fibrous tissue infiltrated by inflammatory cells. The tumor can develop in several organs, such as the brain, spinal cord, larynx, thyroid gland, breast, pancreas, gastrointestinal tract, or urinary bladder, but mostly it develops in the lungs and liver.¹ The pathogenesis of IPT is not clear. Infections (either bacteria or parasites), autoimmunity, radiation, and chemotherapy have been considered as possible causes.² In addition, several diseases have been related to the development of IPT, including Crohn's disease, gastrointestinal stromal tumor, congenital neutropenia, and pregnancy.³ The lesion was first described by Pack and Baker in 1953.⁴

Most patients are in their infantile period and between 40 to 70 years of age, and 60% are female.⁵ The clinical presentation is also variable, including abdominal mass, abdominal discomfort, intermittent fever, weight loss, and general malaise. The laboratory findings may reveal an inflammatory process, with leukocytosis, elevated erythrocyte sedimentation rate, or increased C-reactive protein level. Thus, a clinical diagnosis of IPT is difficult to achieve due to its variable presentation. The treatment is also variable. Traditionally, surgical treatment has been indicated, but recently, spontaneous regression of the tumor has been reported after oral non-steroid anti-inflammatory drugs or antibiotic treatment.⁶

A radiologic diagnosis of IPT is also difficult because it shows variable imaging findings and lacks specific findings. On angiography, the lesion shows hypervascularity, as does that of HCC.⁷ On sonography, the lesion usually appears hypoechoic, but it also can be hyperechoic or lack complex echogenicity. The inner content of the tumor is usually heterogeneous, without definite necrosis or cyst formation.⁸

The image findings on CT are also variable. On precontrast CT, the lesion is usually hypodense, and in the delayed phase the tumor can show a variable enhancement pattern, including homogeneous enhancement, peripheral enhancement, heterogeneous enhancement, or even contrast washout in the delayed phase.⁶ The variable delayed-phase enhancement pattern has been reported to relate to the various components of the fibrotic part of the tumor.⁹ Our CT findings of IPT showed the typical image findings of an HCC enhancing profile, which includes early enhancement, contrast medium washout in the venous phase, and a tumor capsule. The patient had liver cirrhosis, and a preoperative diagnosis of HCC was thus made according to the results of the imaging.

In our case, the tumor capsule seemed thicker than in the usual presentation of HCC. The tumor capsule is an important characteristic of HCC, being between 0.3 mm and 1.4 mm (0.87 ± 0.59 mm) in pathological examination,¹⁰ and 98% of tumor capsules are less than 2 mm thick on an image study.¹¹ However, in our case, the fibrous tumor capsule measured 2–3.3 mm (2.6 ± 0.52 mm), thicker than that seen in typical HCC. Thus, if a tumor presents typical findings of HCC on CT, but has a thicker tumor capsule than usual, IPT should be included in the differential diagnosis, and biopsy should be performed before operation. However, to the best of our knowledge, there is no literature discussing the mechanism of capsular formation in IPT of the liver.

In conclusion, we have presented a case of hepatic IPT that was misdiagnosed as HCC because it presented the typical image findings of HCC on CT. We suggest that a thicker tumor capsule on CT can be an important diagnostic clue to differentiate IPT from HCC.

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